SURFACE ELECTROMYOGRAPHY ACTIVITY IN THE AGONISTS AND ANTAGONISTS OF PRONATION/SUPINATION OF THE UPPER LIMB IN PATIENTS WITH CERVICAL COMPRESSIVE MYELOPATHY

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VELLORE

CERTIFICATE

This is to certify that the work contained in this study entitled "**SURFACE ELECTROMYOGRAPHY ACTIVITY IN THE AGONISTS AND ANTAGONISTS OF PRONATION/SUPINATION OF THE UPPER LIMB IN PATIENTS WITH CERVICAL COMPRESSIVE MYELOPATHY*****" is a bonafide work of Dr. Balaji Srinivas.S, submitted in partial fulfillment for the degree of M.Ch. Neurosurgery (Part III) examination conducted by Dr.MGR Medical University, Chennai in February 2010.***

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CONTENTS

INTRODUCTION

Patients with cervical compressive myelopathy present with dysfunction due to spasticity, weakness and sensory deficits. Relief of the cord compression with surgery results in change in function. The standard methods of assessing clinical status in patients undergoing surgery for cervical compressive myelopathy are the Nurick's grading[1] and the Japanese Orthopaedic Association Scoring system(JOA).[2] However, both these tests are subjective and while the JOA score assesses the upper limbs and lower limbs separately, the Nurick's grading primarily assesses lower limb functions. We earlier described and standardized a test of hand function- the Rapid Hand Flick Test (RHFT) that involves rapid opening and closing of the hand. This was found to be useful in assessing improvement in hand function after surgery for cervical spondylotic myelopathy. During this test, there was a slowing in the opening and closing of the hand preoperatively in patients compared to normal subjects, that improved after spinal decompressive surgery. The patients reported subjective improvement in the tightness in the limbs manifested by improvement in the time taken for 20 hand flicks. Apart from assessing hand function we suggested that the RHFT was an objective measure of spasticity, since co-contraction of the flexors of the forearm would slow down the opening of the hand. We did not have any EMG data to support this hypothesis. In this study, we aimed at studying the surface electromyographic activity from the agonist/antagonist forearm muscles in the upper limbs during alternate pronation/supination and compared the activity with the same post-operatively.

REVIEW OF LITERATURE

Cervical compressive myelopathy is a disabling and distressing neurological disease. The clinical presentation of this entity is diverse. The progressive disability seen in these patients is caused by a combination of muscle spasticity, weakness, and sensory deficits.

Spasticity

Spasticity is a state of increased tone in muscles resulting from hyperactivity of the stretch reflexes. It is defined as a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome.[3] It results from lesions of the pyramidal and often the reticulospinal pathways. With the release from the descending inhibitory influences, the spinal reflexes become hyperactive as a consequence of increased excitability of dynamic motor neurons and alpha motor neurons. In addition, there may be signs such as weakness, impairment of fine motor movements of the digits, loss of cutaneous reflexes, Babinski sign and clonus.

Muscle stretch whether by passive or active limb motion or by tendon tap elicits reflex contractions. Phasic stretch reflexes are elicited by fast but not slow limb motion because the stretch receptors of Ia afferents are velocity sensitive. The reflex muscle contractions fade in seconds because limb velocity slows and afferent discharge rate decreases.[4]

Pathophysiology of spasticity

Spasticity generally develops when suprasegmentary control over the spinal cord segmental reflexes are lost and typically occurs secondary to lesions of premotor areas or their outflow including long spinal tracts. Evidence suggests that the descending tracts may directly modulate not only the afferent limb of the peripheral reflex arcs, but also the anterior horn cells. Spasticity is caused by adaptive changes in transmission in the spinal networks distal to a lesion of descending motor pathways. Selective lesions of the pyramidal tract does not lead to spasticity, whereas lesions of descending pathways from the brainstem as well as the cortical control of these pathways does.[5]

Primary afferent Ia and II fibres surrounding intrafusal fibres of the muscle spindle are excited when a muscle is stretched. The Ia fiber makes a monosynaptic excitatory connection with alpha motor neurons of its muscle of origin and it similarly connects with alpha motor neurons of synergistic muscles. The Ia fibres also monosynaptically connect with inhibitory interneurons that project directly to alpha motor neurons of antagonistic muscles. When a muscle is stretched, excitation of homonymous and synergistic motor neurons combined with inhibition of antagonists, subserves the mechanism of reciprocal inhibition.[4]

Two forms of spasticity are described in the literature

a) Cerebral model

b) Spinal model

Herman et al [6] found that patients with spinal cord lesions had a relatively slow rise of reflex activity compared to cerebral model with peak activity occurring only after a number of stretch cycles were generated. In contrast, Herman notes that in cerebral (or hemiplegic) model of spasticity, sinusoidal stretching of calf muscles results in a rapid build up of reflex activity, suggesting that transmission of primary ending spindle discharges occurs largely through monosynaptic pathways.

The activity of an anterior horn cell is a summation of both excitatory and inhibitory synapses. It includes excitatory post-synaptic potentials from group Ia and II muscle spindle afferents, inhibitory post-synaptic potentials from Golgi tendon organs and antagonistic muscles. It also includes presynaptic inhibition of excitatory sensory afferents synapsing on anterior horn cells through interneuron connections from higher centers of the central nervous system.

There are two possible basic neural mechanisms to explain increased excitability of segmental reflexes:

1. Selective increase in motor neuron excitability

2. Increase in amount of excitatory potentials generated from muscle stretch receptors

There is experimental evidence from animal models of spasticity for motor neuron excitability. The etiology of this phenomenon could be caused by extrinsic factors acting on motor neurons or intrinsic changes in motor neuron pushing them closer to the threshold. There is some evidence for enhanced extrinsic excitatory inputs (from segmental afferents, regional excitatory interneurons, and descending pathways such as reticulospinal and lateral vestibulospinal tracts) and greater

support for diminished extrinsic inhibitory inputs on the motor neurons (from regional inhibitory interneurons, such as Renshaw autogenic inhibition, Ia inhibitory interneurons, or Ib afferents). So far, there has not been evidence to support intrinsic changes in the motor neuron cells.[7]

Some studies also suggest that increased stretch evoked synaptic excitation of motor neurons contributes to the clinical presence of spasticity. Possible physiologic mechanisms to explain this phenomenon are hyperactivity of the gamma loop or excitatory interneurons becoming more sensitive to inputs from muscle afferents.[8]

Three possible mechanisms have been postulated for increased sensitivity of excitatory interneurons:

1. collateral sprouting
2. denervation hypersensitivity
3. diminished pre-synaptic inhibition

In spinal cord lesions, impaired transmission in different spinal inhibitory pathways, such as reciprocal inhibition and presynaptic inhibition, has been found which probably contributes to exaggeration of reflex activities and increased muscle tone.

Wiesendanger et al [8] stated that spasticity "is characterized by altered activity patterns of motor units occurring in response to sensory and central command signals which lead to co-contractions, mass movements and abnormal postural control". McLellan et al [9] suggested "excessive and inappropriate muscular activation

occurring in association with the upper motor neuron syndrome" as a functional definition of spasticity. Regardless of the mechanism (loss of inhibition, changed properties) motoneuron hyperexcitability is centrally involved in all these manifestations of spasticity.

Normal tone consists of a balance between inhibitory effects on stretch reflexes mediated by the dorsal reticulospinal tract and facilitatory effects on extensor tone, mediated by the medial reticulospinal tract, and, to a lesser extent in humans, by the vestibulospinal tract. In cortical and capsular lesions some of the drive on the inhibitory centre in the caudal brainstem is lost resulting in a spastic hemiplegia, in which antigravity posture predominates, but flexor spasms are unusual. In practice, partial spinal lesions usually involve the lateral corticospinal and dorsal reticulospinal tract.[10]

Damage to the corticospinal tract leads to paresis, while loss of inhibitory influences from the dorsal reticulospinal tract, leaves the effects of the medial reticulospinal and vestibulospinal tracts unopposed. In this situation there is often severe spasticity with tone being greatest in the antigravity muscles (flexors and pronators of upper and extensors and adductors of lower limbs) so that paraplegia in extension may be seen. Extensor and flexor spasms are common, although the former tend to predominate. There is more passive resistance to extension than to flexion in the upper extremities, and to flexion than to extension in the lower extremities.[10]

In severe or complete cord lesions there is loss of all supraspinal influence on the cord. Hypertonicity is not as marked as in some cases of incomplete cord lesions, as the descending excitatory systems are no longer acting unopposed.

Flexion spasms are very prominent, however, as flexor reflexes are released from the inhibitory influences of the dorsal reticulospinal, vestibulospinal and medial reticulospinal tracts. Paraplegia in flexion may then supervene. The pattern of spasticity is not fixed and solely determined by the degree of damage to different descending pathways. Stimulation of flexor reflex afferents-for example, by pressure sores, can transform paraplegia in extension into paraplegia in flexion. Conversely, standing reduces flexor tone and favours extensor tone, a phenomenon that is readily used to advantage in physiotherapy. An additional factor in complete transection is the delayed reorganisation within the isolated cord, which may underline the change in balance from flexor to extensor spasms sometimes seen a year or more after division of the cord.[10]

Spastic co-contraction

Co-contraction refers to the simultaneous contraction of both agonist and antagonist muscles. In normal postnatal motor development, extensive co-contraction is a normal feature, associated with heteronymous, monosynaptic Ia projections from biceps to triceps and to regional synergists and antagonists.[11] Normally these connections become restricted to primary synergists in the 4 years of life. Co-contraction is dysfunctional when it is inappropriate or excessive and impairs agonist function, also making the agonist appear weaker than it is. The question of the existence and importance of co-contraction has therapeutic relevance; inappropriate antagonist contraction could be reduced focally by botulinum toxin injections [12] or phenol nerve blocks or by other antispasticity agents such as baclofen.[13] Patients with spastic disorders often un-intendedly co-contract antagonist muscles during various muscles such as gait. This co-contraction occurs

at inappropriate times and can therefore be a major obstacle for the movement.[14] During voluntary movement in healthy subjects, the activity of antagonistic muscles is controlled by central modulation of the transmission in the inhibitory pathways, which link the muscles.[14] It is a likely possibility that a deficient control of these inhibitory mechanisms is, at least partly, the basis of the inappropriate antagonistic co-contraction in spastic patients. The pathophysiological substrate of cocontraction is impairment of Ia reciprocal inhibition [14,15] in the spinal cord. The di-synaptic Ia inhibitory pathway is the most thoroughly studied inhibitory spinal pathway. The activity in the pathway has been studied in man by making use of the Hoffman refex (H-reflex) technique. In these experiments, the H-reflex of one muscle is conditioned by a preceding stimulation of the nerve to the antagonistic muscle. The disynaptic inhibition is thereby revealed as a depression of the H-reflex 1-3 milli seconds following the conditioning stimulation.

In a study by Berardelli et al [15] which was performed on 47 patients with varied etiology (multiple sclerosis, cerebral palsy, stroke, cervical spondylosis, cortical tumours, amyotrophic lateral sclerosis, primary lateral sclerosis, chronic myelopathy and olivo-ponto-cerebellar degeneration), the authors showed a clear relationship between the tendon jerk and the short-latency reflex magnitude and rate. The short-latency reflex is more objective than the routine clinical measure and would be a useful parameter in any quantitative assessment of the phasic stretch response of spastic patients. From their data they concluded that long-latency reflex behaviour certainly also makes a contribution to spastic tone. Some patients showed long-latency reflexes which are increased in magnitude and others showed long-latency reflexes increased in duration and both of these phenomena correlate with the clinical impression of tone. They showed that there is more than one type of

spastic tone that do not have a homogeneous aetiology. They also postulated that there are multiple mechanisms for the long latency reflexes some of which may only appear in pathological states.

Daofen Chen et al [16] evaluated the possibility that descending systems have differential actions on the spinal interneurons that receive input from muscle afferents. Prolonged, physiological inputs were generated by stretch of the triceps surae muscles. The resulting firing patterns of 25 lumbosacral interneurons were recorded before and during a reversible cold block of the dorsolateral white matter at the thoracic level in nonparalyzed, decerebrate preparations. The strength of group I muscle afferent input was assessed from the response to sinusoidal tendon vibration, which activated muscle spindle Ia afferents directly and tendon organ Ib afferents via the resulting reflex force. The stretch evoked responses of interneurons with strong responses to vibration were markedly suppressed by dorsal cold block, whereas the stretch evoked responses of interneurons with weak vibration input were enhanced. The cells most strongly activated by vibration received their primary input from Ia afferents and all of these cells were inhibited by the cold block. These results suggest that a disruption of the descending system, such as occurs in spinal cord injury, will lead to a suppression of the interneuronal pathways with group Ia input while enhancing excitability within interneuronal pathways transmitting actions from higher threshold afferents. One possible consequence of this suppression would be a decreased activity among the Ia inhibitory interneurons that mediate reciprocal inhibition, resulting in abnormal reciprocal relations between antagonists and promoting anomalous muscle cocontraction. The authors[15] found that interneurons with a strong group I input are preferentially facilitated by tonically active pathways descending in the dorsolateral quadrants, while interneurons with

weak group I input are inhibited. These tonically active pathways include the dorsal reticulospinal tract and may also include the rubrospinal tract and long propriospinal tracts [17]. Vestibulospinal inputs excite Ia inhibitory interneurons [18]. They did not exclude the possibility that some of the interneurons could have been ascending tract cells. However, the cells classified as Ia, II, or Ib interneurons are probably not ascending tract cells, or receive only relatively weak proprioceptive input.[18] The differences between sustained physiological inputs versus transient inputs may be important for descending control of reciprocal inhibition. As a result, it is conceivable that much of the normal reciprocal relations between antagonist muscles could be lost, promoting inappropriate muscular cocontraction. These changes may seriously impede efforts at restoring locomotor patterns, which require strong reciprocal relations between antagonists.[19]

Normally, agonist Ia activity exerts an inhibitory effect on the antagonist motoneurones via an interneurone. This activity is influenced by supraspinal inputs and by other segmental afferents.[20] Abnormalities of Ia reciprocal inhibition have been reported in spasticity and could contribute to co-contraction.[14] Impaired spinal Ia reciprocal inhibition probably arises from disordered supraspinal modulation. In spastic cerebral palsy the heteronymous Ia connections of infancy mentioned earlier are persistent, but this is not the case in adult hemiparesis due to stroke.[11]

Wiesendanger et al [8]pointed out the necessity to understand tone as a part of postural control, and that it must have prospective as well as reactive components. Ultimately, such control is the expression of the brain. Spasticity and related upper motor neuron dysfunction should be recognized as a motor behavior. Thus, to control

the variability, both behavior of the subject and external stimuli (acoustic, etc.) must be controlled .[9]

Clinical assessment of spasticity

The quantification of spasticity has been a difficult and challenging problem and has been based primarily on highly observer-dependant measurements. The lack of effective measurement techniques has been quite restrictive, since quantification is necessary to evaluate various modes of treatment.[21] Assessment of spasticity includes identifying which muscles or muscle groups are overactive and determining the effect of spasticity on all aspects of patient function, including mobility, employment, and activities of daily living (ADLs). Spasticity is generally manifested by increased muscle tone during movement. Abnormalities of muscle tone can be clinically assessed in the upper limbs by supination and pronation at the elbow.

For clinical assessment of spasticity, the Ashworth and modified Ashworth scales are commonly used. A clinical scale from 0 (normal muscle tone) to 4 (severe spasticity) was first proposed by Ashworth.[22] The Ashworth scale suffers from clustering of most patients within the middle grades. It offers ease of measurement, but may lack temporal and inter-examiner reproducibility. The Modified Ashworth Scale (MAS) by Smith and Bohannan [23]is extended with an extra grade between 1 and 2 (i.e., 1+). The scores are determined by moving the joint over its entire range of motion. These scales provide a semiquantitative measure of the resistance to passive movement, but have limited inter-rater reliability.[21] Clinical scales offer only qualitative information, but they are the most widely used yardstick of spasticity.

In a previous study we compared a new hand function test described by us [24] called the rapid hand flick test (RHFT) with the Jebsen-Taylor test of hand function. The Jebsen-Taylor test of hand function[25] assesses hand disability and improvement in hand function after therapeutic intervention. In the above study, one hundred normal subjects and 26 consecutive patients undergoing surgery for cervical spondylotic myelopathy were studied. Complete, rapid opening and closing of the hand was timed for 20, 40, and 60 repetitions called as The Rapid Hand Flick Time, preoperatively and at the end of the first week postoperatively. The results of this test were correlated with the Jebsen-Taylor test. There was a 40% to 50% prolongation in the time taken for the rapid hand flick in patients compared with age-matched normal subjects. Postoperatively, there was a 3.84-second mean improvement in the RHFT for 20 repetitions in the right hand and 2.8 seconds in the left hand.

A paired-samples t test, comparing the preoperative and postoperative day 1 and day 7 timings, showed a statistically significant improvement (P=.001). Improvement in the RHFT20 and Jebsen-Taylor test in the early postoperative period was postulated to be an objective representation of the subjective sense of neurologic improvement reported by most patients postsurgery. The test was concluded to be a reliable and reproducible bedside test of hand function in the immediate postoperative period. After decompressive surgery, early improvement in hand function related to improvement in muscle power and spasticity was expected to occur, because some of the intramedullary pathologic changes in cervical spondylotic myelopathy are reversible in the short term.

Electrophysiological testing

Surface electromyography (EMG) is used to estimate muscle activation. It entails using surface electrodes which are non-invasive and, because they have a large pick up area, they are appropriate for the study of gross muscle function. They are well suited for the study of the temporal relationship between the EMG signal and muscle contraction dynamics and, to a limited extent, the magnitude of muscle contraction force.[26,27] The use of surface EMG makes it possible to examine the behavior of a large set of muscles in each subject.[26,27]

In a study by Van der Selm et al [28] in 9 patients with complete spinal cord injury, the authors developed a method for assessment of spasticity in which the whole range of motion at a wide variation of speeds was applied. The patients were seated upright and the knee was flexed. The foot was fixed to a footplate, which could be rotated around one axis, thus providing plantar flexion and dorsiflexion at the ankle joint. By using a handle, dorsiflexion and plantar flexion movements were applied manually by the investigator. The movement of interest was the dorsiflexion movement to assess soleus muscle spasticity, which is clinically most relevant. The electrodes were placed on the soleus muscles and not on the anterior tibial muscles. The stretch movements were applied manually, comparable to the stretches of MAS movements and stretches in daily life. The electromyographic responses were measured during dorsiflexion at the ankle by applying 30-45 stretches over the whole range of motion at varying velocities from 30-150 degree/sec. They detected the start of the electromyographic activity during the stretch and the root-mean-square (RMS) values at three speeds (50-150 degree/second) were analysed. The resistance to passive movement over the ankle joint was measured as torque with a calibrated

strain gauge during the whole stretch. The ankle angle was measured using a potentiometer at the axis of rotation and the angular velocity was determined with a gyroscope fitted on the footplate. When the stretch velocity increases, more I-a afferents from the muscle spindles will be recruited. After reaching a certain threshold the alpha motoneurons generate action potentials activating the innervated muscles. When the velocity is continuously increased, the amount of participating monosynaptic reflexes increases simultaneously. In addition, bisynaptic and polysynaptic reflexes will be activated, initiated by multiple sensory systems (muscle spindles, tendon organs, skin, joint and ligament receptors). This recruitment occurs gradually. At a certain stretch velocity, all reflexes will be recruited.[29]

The authors (Van der Selm et al)[28] found that the electromyographic responses (amplitudes) increased significantly at increasing stretch velocities.($p<0.05$). They showed that movement velocity influences the magnitude of the EMG response and the reflex activity was initiated at specific ankle angles, independent of the stretch velocities. There is no specific mention of co-contraction being studied in this study. They concluded that the method and the device they described could objectively assess muscle spasticity and the stretches applied in their system was comparable to the stretches occurring in daily life. There were several advantages of manually performing the testing over a motorized device. The setup would be less complex, less expensive, more applicable in a clinical setting and that clinicians would be able to feel the movement.

In a study by Skold et al[30,31] the authors investigated whether the modified Ashworth scale (MAS) was a valid measure of spasticity in motor-complete quadriplegic spinal cord injured individuals. To evaluate the usefulness of the

subjective Ashworth measurements in these patients, they performed simultaneous EMG recordings in the knee flexors and extensors during flexion and extension, and the degree of correlation between the Ashworth measurements and EMG recordings (amplitudes) was analysed. In 15 of 38 randomly selected individuals with complete spinal traumatic quadriplegia, the authors proposed to compare simultaneously performed clinical and neurophysiologic tests as they had not been reported before. They did not evaluate patients with incomplete spinal cord injury. They selected motor-complete quadriplegic patients because they felt when spasticity is elicited by a provoking movement, it is difficult to judge whether voluntary motor function is added as an antagonist or agonist activation. In this study, spasticity evaluation was done by passively flexing and extending the knees with the subject lying in the supine position, with the knee joints at the edge of the bench. The lower leg was moved by grabbing the subject's ankle with one hand and stabilizing the distal femur during both movements. Simultaneous EMG recordings of quadriceps (knee flexors) and hamstrings (knee extensors) and modified Ashworth Score (MAS) assessment was done. The knee was flexed and extended once during simultaneous EMG registration. The testing was done on both the sides. The EMG baseline was defined as mean electrical activity before and after the movement-associated electrical activity during the 10-second recording period. The activity on the EMG including peak (highest voltage after 1 to 3 second from the start of EMG registration), duration of continuous electrical activity and the start of the electrical activity were recorded. Simultaneous recording of antagonist and agonist muscles was made during flexion and extension movements of the knee. The following components of the muscle contraction were chosen a basis for correlations between Ashworth grade and EMG activity: agonist and antagonist individually, net antagonist (difference between the

agonist and antagonist electrical activity), agonist and antagonist summation. For each of these measurements, they found that the correlation was significant ($p<0.001$) for the antagonist, summation and net antagonist activity on the right side than on the left side. When comparing left/right differences in mean EMG electrical activity, the left side consistently showed shorter duration and lower mean electrical activity irrespective of movement. The left side showed lower peak electrical activity for extension compared to the right side. Mean Ashworth values were lower for left side regardless of movement. The patients in this study were quadriplegic, and there is no mention of the differences in the power and bulk of different muscles since there was a difference in the activity noted here. The right/left difference was proportionally similar for both Ashworth and EMG values. They found that there was a positive correlation between the Ashworth measurements and the EMG parameters such as the amplitude and the duration of contraction in the knee flexor and extensor. The amplitude of contraction and duration was higher as the Ashworth scores increased. During EMG recording, coactivation was always found in the antagonistic muscle groups in both movements. They measured agonist and antagonist activity individually, net antagonist (difference between the agonist and antagonist electrical activity), agonist and antagonist summation. Spearman correlation coefficient was applied and they found a statistically significant correlation ($p<0.05$) between the Ashworth scores and the EMG measurements. Extension showed a better correlation between Ashworth method and EMG recordings than flexion. The duration of movement associated electrical activity and the mean electrical activity was higher during extension than flexion movements. They felt that the difference was due to different amounts of subcutaneous fat tissue between the electrode and the tested muscle and actual mass of the muscles. They

also considered that due to the larger range of motion of extension than flexion during this test, there was longer duration of movement-associated electrical activity for the extension. The extension movement reached the end of range of motion for the hamstrings compared to flexion, which only reached slightly past the middle of the quadriceps range of motion.

They opined that a grade in the Ashworth scale is a subjective integration of complex muscle activity during the movement. The movement-provoked muscle activity rated on the MAS significantly positively correlated with the amplitude (mean, peak and start-peak) and duration of activity ($p<0.05$). They finally concluded that all included EMG parameters were significantly correlated with simultaneous Ashworth measurements of spastic muscle contraction.

Engsberg et al[32] studied a single patient who underwent C2-3 anterior cervical discoidectomy and fusion for cervical spondylotic myelopathy with biomechanical and electrophysiological measures. The spasticity was assessed at the ankle (plantar flexion and dorsiflexion) and elbow (flexion and extension) bilaterally with the use of a KinCom isokinetic dynamometer. The tests were conducted at speeds of 10, 30, 60, 90 and 120 degree/second. The subject sat on the KinCom dynamometer and had the ankle (or elbow) joint axis aligned with the center of the KinCom lever arm.

The range of motion limits for both the joints were established. The subject was instructed not to assist or resist the lever arm as it moved. The areas within the torque-angle curves were calculated for each speed of the subject. The work values were determined which represented the amount of work required by the KinCom to move the passive ankle or elbow throughout its range of motion at each speed. The

slope of the linear regression line was considered to be the magnitude of the spasticity. A slope near zero represented no spasticity, whereas increasing slopes represented increasing amounts of spasticity. They found that there was reduction of elbow flexor spasticity from 0.04 joules/degree/second preoperatively to 0.00 joules/degree/second postoperatively at 11 days. There were less significant results obtained for the ankle dorsiflexors and plantar flexors. The most dramatic change was the improvement in walking speed, from 73 cm/second before surgery to 82.6 cm/second to 100.3 cm/second 6 months after surgery. The drawback of this study was that with only one subject, it is difficult to predict whether the change will be characteristic of a similar group of patients. The exact parameters measured and also the parameters in able bodied individuals as mentioned in the study are not clear. However it laid a basis for the use of biomechanical methods to measure changes in function and impairments associated with surgical intervention for cervical spine disorders.

With the previous studies on lower limbs for the assessment of spasticity with surface EMG activity, we proposed to utilize this combination of clinical and electromyographic recordings to study our patients with upper limb spasticity. These are the patients who undergo decompressive surgery and since there are no previous such studies, we proposed to observe in them the changes that occur before and after cervical decompressive surgery.

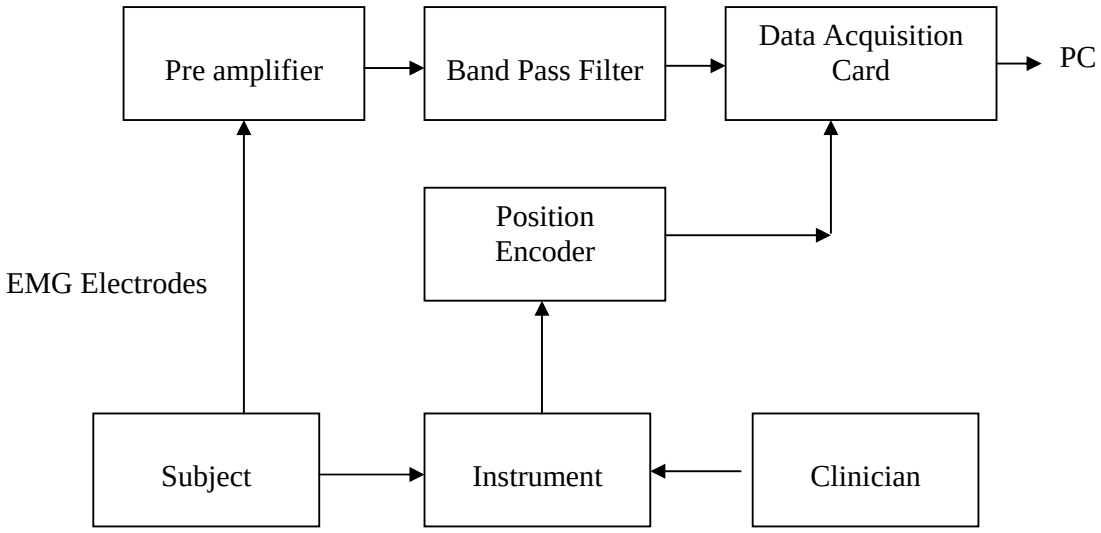
AIMS AND OBJECTIVES

1. To study the surface electromyography activity in the agonists and antagonists of pronation/supination in the upper limbs in patients with cervical compressive myelopathy .

2. To compare the preoperative and immediate post-operative surface electromyography activity in patients with cervical compressive myelopathy undergoing surgery.

MATERIAL AND METHODS

DESCRIPTION OF THE APPARATUS AND THE TEST



During the detection of the surface electromyography recordings from the forearm of these patients, the patient is seated comfortably on a chair with the elbow supported in a holder attached to the chair. The skin of forearm and arm are prepared with alcohol (70%) to decrease the impedence which would affect the recording. Surface electrodes (Discoid, Ag-AgCl gel) with a diameter of 10 mm are placed with an inter electrode distance of 10 mm as follows: for the biceps brachii at 1/3 of the length from the cubital fossa to the acromion process and for the pronator teres 3 cm distal to the midpoint of a line connecting the medial epicondyle of the humerus and the biceps tendon. The reference electrode is placed between the two recording electrodes against the medial epicondyle. The surface electromyographic activity is measured from the pronator teres and biceps brachii while pronation-supination is performed actively by the patient and then passively by the investigator who rotates the device thus creating a pronation-supination movement. When clinically assessing these patients, increasing velocities were used for assessing spasticity till a resistance or 'catch' is felt, and a comparable range of velocity was used by the investigator during the experimental recording of the surface electromyographic activity.

The resistance felt to the passive movement is a subjective assessment by the clinician and was noted as the modified Ashworth score. The following data were collected from the electromyographic recordings initially during active movement (where the subject moves the device) and then passive movement (where the device and the patient's forearm was moved by the clinician) in the pronators and supinators

- Surface EMG activity (amplitude of the RMS EMG activity)

- Range and velocity of movement.

The EMG signals from the electrodes were amplified (666x) using a high precision instrumentation amplifier. The amplified signal was passed through a band pass filter (15, 1.5K) Hz to reduce the movement artifact and other noise influences as per standards. The range of movement was encoded using an optical disc encoder with a resolution of 8 degrees. The analog signal of EMG activity was fed to the personal computer through a data acquisition card (National Instruments) at 1000 samples with resolution of 14 bit conversion.

To record the EMG and position signal, a custom graphical user interface was made. Similarly, to review the recorded signal another graphical user interface was made using Labview 8 (National Instruments).

The neutral position for the test was the fully supinated forearm. The midway position between pronation and supination was not considered to make both the movements equal. The direction sensor in the device gave information about the movement- a positive deflection denoting pronation and a reversal of the deflection to the negative side denoting supination. The position encoder provided the information about the start and end of each movement. Each pulse of the encoder was eight degrees and the range of movement was thus calculated from the number of pulses and velocity was given by the time taken for the complete range of motion. Three trials were taken and an average value was calculated for the analysis.

The review program gave the plot of root mean square (RMS) EMG activity, raw EMG activity, range of movement and angular velocity.

ASSESSMENT OF THE PRONATION/SUPINATION AND THE SURFACE EMG

NORMATIVE DATA

We collected normative data in 31 age related subjects (25 males and 6 females) with a mean age of 53.39 years(range 29-62) by administering the test using the above described pronation-supination apparatus.

STUDY POPULATION

Patients with cervical compressive myelopathy undergoing decompressive surgery were included in the study**.** It was approved and supported by grants from the institutional Research Board (Fluid Research Committee, IRB Min No. 6698). Patients were enrolled after obtaining informed consent.

Inclusion Criteria:

- Patients who were 18 years and above with cervical compressive myelopathy
- presence of spasticity >grade 1 (Modified Ashworth Score)
- Nuricks grade 1-4

Exclusion Criteria:

- patients with lower motor neuron findings in the upper limbs on clinical examination
- patients who were Nurick grade 5, bed bound and difficult to transfer for the test to the Neurophysiology laboratory.
- Patients with joint deformities, contracture, thrombophlebitis,

The group included 31 non consecutive patients: 3 were females and 28 were males. The incidence of cervical spondylotic myelopathy in females is markedly less than males in our population. The mean age was 54.06 years (range 25-72 years). All patients underwent a detailed neurological evaluation preoperatively and postoperatively. Their pre-operative functional status was assessed by the Nurick grade and Japanese Orthopaedic Association (JOA) Score. Modified Ashworth Scores (MAS) were determined for the subjective assessment of spasticity.

The pronation/supination test was administered to the patients pre-operatively and one week after undergoing a decompressive surgery.

STATISTICAL ANALYSIS

The SPSS 16.0 software package was utilized to analyze the data. All values are expressed as means ± standard deviation. We calculated descriptive statistics for continuous variables and obtained frequency distributions for continuous variables. We used the independent sample student t-test to calculate the significance of the association for continuous variables between groups and employed the paired t-test to compare pre and post surgical assessments in the patient group. We used Pearson correlation coefficient to assess the significance of associations. Mean and median were calculated based on the distribution of parameters.

A p value <0.05 was taken as statistically significant

A p value of <0.001 was taken as highly significant

RESULTS

Normative data :

The data from the age adjusted 31 controls (25 males and 6 females) with a mean age of 53.39 (+/-8.6) years (range 29-62) showed the assessment on the various scales (Nurick, JOA, MAS) was normal as they had no complaints and did not have any neurological deficits.

Study population :

Pre-operative clinical and demographic profile:

There were 31 patients in the study; of these 28 (90.3%) were males and 3 (9.7%) were females. The age ranged from 25 to 72 yrs. (mean age of 54.06 yrs± 10.12sd). Most of the patients were in the age group of 51-60 years. The most common etiology of cervical compressive myelopathy in our patients was cervical spondylotic myelopathy. Other etiologies were intervertebral disc prolapse (IVDP) and craniovertebral junction (CVJ) anomaly (Table 1).The duration of symptoms in the patients ranged from less than 6 months upto 5 years. There were 18 patients who were symptomatic for less than 6 months. Two patients had longstanding symptoms for more than 4 years. None of the patients in our study had contractures (Table 2). Most of the patients underwent anterior cervical discoidectomy, corpectomy (oblique or central) or laminectomy for cervical spondylotic myelopathy. Three patients underwent instrumented fusion for craniovertebral junction anomalies (Table 3). The clinical assessment and the various scores on Nurick, JOA and MAS scales of the patients are shown in Table 4-7.

Post-operative clinical profile:

13 patients out of 31 had a MAS score of 2. Among the 31 patients, 14 patients with MAS scores of 2 and 3 improved by 1 score. Only patient with a score of 4 improved post-operatively by a score of 2. The mean MAS score reduced from 1.97(0.84) preoperatively to 1.45(0.51) postoperatively (p value 0.000) (Table 4 a,b). It can be seen in the Fig. 4 b that all the numbers along the diagonal i.e., 17 patients remained the same post-operatively in the MAS score, and those to the left of the diagonal improved in their MAS score -13 patients by 1 grade and 1 patient by 2 grades.

13 out of 31 patients had an improvement in their upper limb JOA score (11 by 1 score and 2 patients by 2 score). Those who had improvement had a pre-operative score of 4-6. The mean JOA (upper limb) score improved from 5.45(1.26) preoperatively to 5.94(1.03) postoperatively (p value 0.000) (Table 5 a,b). It can be seen in the Fig. 5 b that all the numbers along the diagonal i.e., 18 patients remained the same post-operatively in the JOA score, and those to the left of the diagonal improved in their MAS score -11 patients by 1 grade and 2 patients by 2 grades.

15 patients out of 31 were of Nurick grade 4. Those patients with a Nurick grade of 2 and 3 remained the same, but there was an improvement by 1 grade in patients with Nurick grade 4 and 5 (2 in each). The mean Nurick grade reduced from 3.61(0.88) preoperatively to 3.48(0.81) postoperatively (p value 0.043) (Table 7). It can be seen from Table 6 that before and after surgery, there was statistically significant change in the MAS and JOA scores (p value <0.05).

Table 1. Distribution of patients according to Age and Sex

Age group (yrs)	Male	Female	Total	Diagnosis
21-30	0	1	1	CVJ anomaly
31-40	2	0	2	1 CVJ anomaly, 1 IVDP
41-50	6	2	8	1 CVJ anomaly , 3 CSM, 4 IVDP
51-60	10	0	10	7 CSM, 3 IVDP
61-70	9	0	9	7 CSM, 2 IVDP
71-80	1	0	1	IVDP
Total	**28**	**3**	**31**	

Table 2. Distribution of patients according to duration of symptoms .

Duration(in months)	Number of patients (n=31)
0-6	18
7-12	6
13-24	3
25-48	2
49-60	2

Table 3. Distribution of patients according to the surgical procedure

Surgical procedure	Number of patients(n=31)
Anterior cervical discoidectomy (single level)	12
Anterior cervical discoidectomy (more than one level)	2
Central corpectomy	1
Oblique corpectomy	10
Cervical laminectomy	3
Instrumented fusion for CVJ anomaly	3

Table 4(a).Distribution of patients according to pre and post- operative modified Ashworth (MAS) score

MAS score (Upper limb)	MAS score (Upper limb) Post-op			No.of patients (N=31)
Pre-op	Same N=17	Improved (by 1grade) N=13	Improved (by 2 grade) N=1	
1	10	0	0	10
2	7	6	0	13
3	0	7	0	7
4	0	0	1	1

Table 4(b).Distribution of patients according to MAS score

		Post-operative MAS score			
		1	2	3	4
	n=31	16	15	0	0
Pre-operative MAS score 1	10	10			
2	13	6	7		
3	7		7	0	
4	1		1		0

Table 5(a).Distribution of patients according to pre and post-operative upper limb JOA score

JOA score (Upper limb)	JOA score (Upper limb) Post-op			No.of patients (N=31)
Pre-op	Same N=18	Improved (by 1grade) N=11	Improved (by 2 grade) N=2	
4	2	6	2	10
5	4	2	0	6
6	4	3	0	7
7	7	0	0	7
8	1	0	0	1

Table 5(b).Distribution of patients according to pre and post-operative upper limb JOA score

Post-operative upper limb JOA score

Pre-operative upper limb JOA score

		8	7	6	5	4
	n=31	1	10	8	10	2
8	1	1				
7	7		7			
6	7		3	4		
5	6			2	4	
4	10			2	6	2

Variable	Pre-surgical assessment Mean (sd)	Post-surgical assessment Mean (sd)	p value
Nurick score	3.61 (0.88)	3.48 (0.81)	0.043
MAS	1.97 (0.84)	1.45 (0.51)	0.000
JOA upper limb score	5.45 (1.26)	5.94 (1.03)	0.000

Table 6. Pre and post-operative clinical variables with the significance

Table 7.Distribution of patients according to Nurick grade

Nurick grade (Preop)	No. of patients (Pre-op)	No. of patients (Post-op)
2	4	4
3	8	10
4	15	15
5	4	2

Surface EMG recordings in controls and patients:

The baseline activity in the pronators and supinators was less in the controls than the patient population. It was 0.031(0.011) mV in controls and 0.069(0.025) mV in the patients in the pronators (p=0.000). The mean baseline activity in the supinators in the controls was 0.015(0.007) mV and 0.076(0.078) mV in the patients. (p=0.000).

Table 8 and 9 shows the mean RMS amplitude in controls during active and passive pronation and supination in the pronators and supinators.

Table 8. Mean RMS amplitude (in mV) in 31 controls in pronators and supinators during active movement

N=31	Mean RMS amplitude (in mV)	+/-sd
Pronators during pronation	0.602	0.230
Supinators during Pronation	0.023	0.010
Supinators during Supination	0.430	0.380
Pronators during Supination	0.035	0,015

Table 9. Mean RMS amplitude(in mV) in 31 controls in pronators and supinators during passive movement

N=31	Mean RMS amplitude (in mV)	+/_sd.
Pronators during pronation	0.035	0.011
Supinators during pronation	0.160	0.051
Supinators during supination	0.018	0.005
Pronators during supination	0.251	0.104

Table 10 shows the mean velocity and range of movement during active and passive pronation and supination.

Table10. Mean velocity (in degree/sec) and range of movement (in degree) in 31 controls during pronation and supination

N=31	Mean velocity (in degree/sec)(sd)	Mean range of movement (in degree) (sd)
During pronation (active)	487.68(91.91)	139.16(13.95)
During supination (active)	414.97(104.89)	139.68(14.89)
During supination (passive)	454.68(55.13)	148.45(10.48)
During pronation (passive)	452.81(55.15)	146.68(9.32)

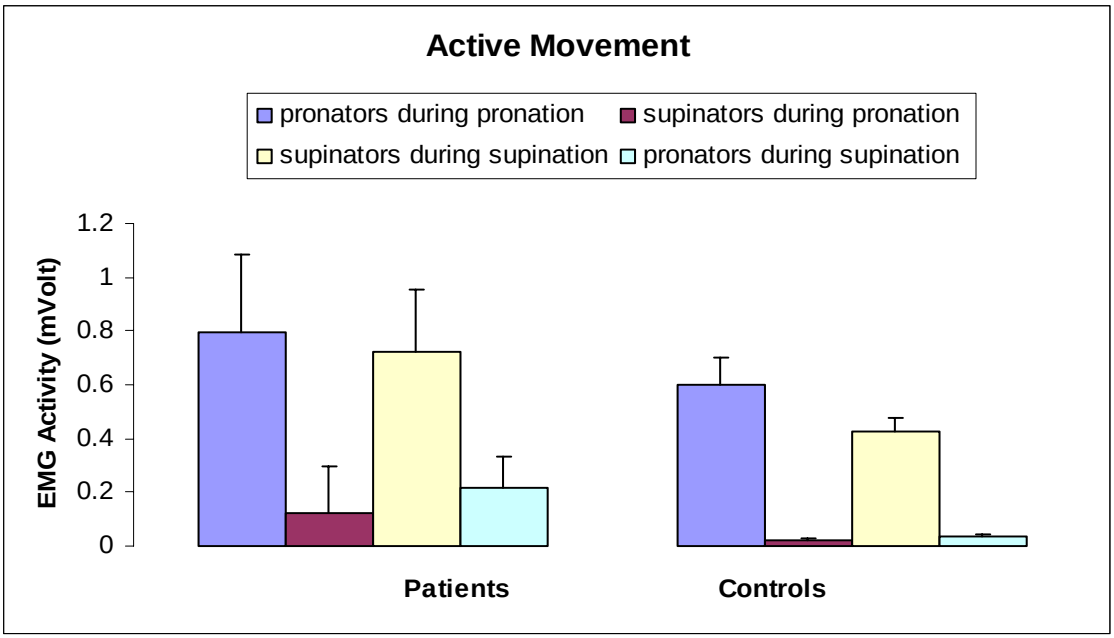
Active pronation and supination in controls and patents:

Table 11 compares the mean RMS amplitude in the patients and controls during active pronation and supination. It can be seen that the EMG activity generated during active movement in the agonist muscle was consistently greater in the patients compared with the controls. For instance, it was 0.795 mV and 0.602 mV in the pronators during pronation in the patients and controls respectively. It was 0.721 mV and 0.430 mV in the supinators during supination in the patients and controls respectively. This was statistically significant for the supinators (p=0.008) but just missed significance for the pronators (p=0.052). On analyzing the antagonistic muscles, it can be seen that the activity in these muscles was significantly greater in the patients as compared to that in the controls. For instance, during pronation the activity in the supinators was 0.125 mV for the patients whereas it was only 0.023 mV in the controls (p=0.000). Similarly , during supination pronator activity was found to be 0.219 mV in the patients , but only 0.035 mV in the controls (p=0.000). This is graphically depicted in Fig. A

Table 11. Mean RMS amplitude in 31 controls and 31 patients in pronators and supinators during active pronation and supination

Movement	Muscle	Mean RMS amplitude (mV) (sd)		P value
		Patients	Controls	
Pronation	Pronators	0.795+/_0.487	0.602+/_0.230	**0.052**
	Supinators	0.125+/_0.088	0.023+/_0.010	**0.000**
Supination	Pronators	0.219+/_0.161	0.035+/_0.015	**0.000**
	Supinators	0.721+/_0.45	0.430+/_0.38	**0.008**

Fig A. EMG activity (amplitude in mV) in 31 controls and 31 patients during active movement



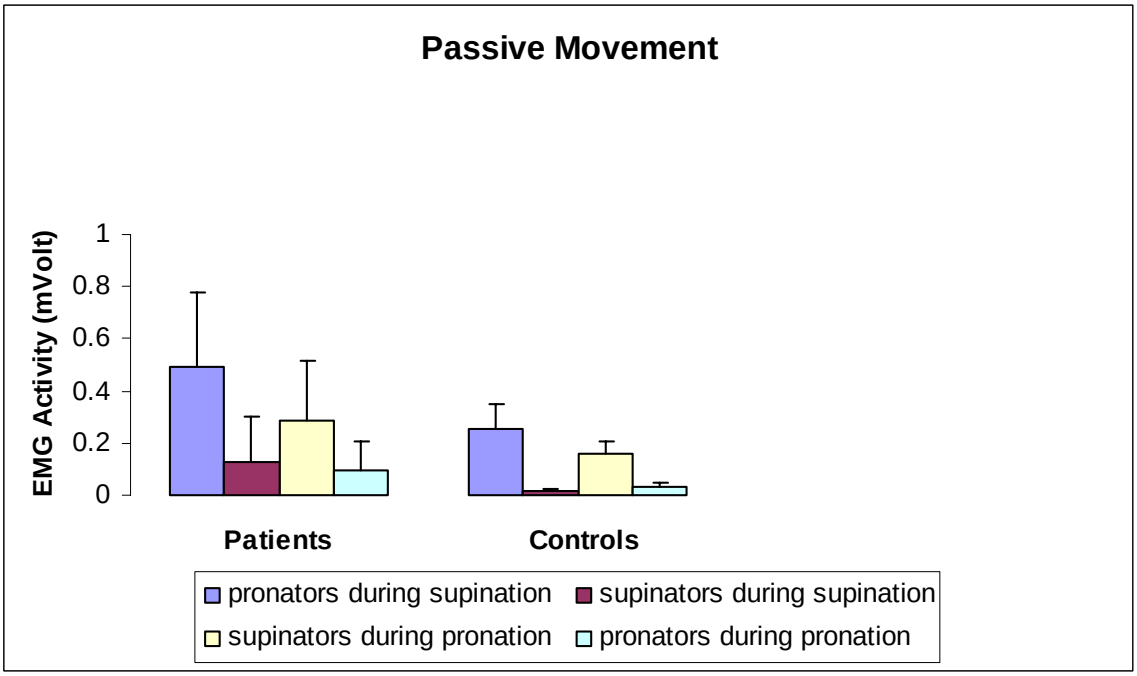
Passive pronation and supination in controls and patients:

Table 12 shows the mean RMS amplitude in the patients and controls during passive pronation and supination. The mean(sd) peak amplitude of contraction during passive pronation in the pronators was 0.097(0.11) mV and supinators was 0.284(0.23) mV in the controls and it was 0.035(0.011) mV in the pronators and 0.160(0.051) mV in the supinators in the patients. It can be seen from Table 12 that during passive movements, the co-contraction in the muscle being stretched was significantly greater in the patients as compared to controls. This was particularly noticeable during supination where the pronator activity was 100% increased (0.492 mV) in the patients as compared to 0.251 mV in the controls (p=0.000). This is graphically depicted in Fig. B.

Table 12. Mean RMS amplitude in 31 controls and 31 patients in pronators and supinators during passive pronation and supination

Movement	Muscle	Mean RMS amplitude (mV) (sd)		P value
		Patients	Controls	
Pronation	Pronators	0.097+/_0.11	0.035+/_0.011	**0.003**
	Supinators	0.284+/_0.233	0.16+/_0.05	**0.005**
Supination	Pronators	0.492+/_0.297	0.251+/_0.104	**0.000**
	Supinators	0.130+/_0.179	0.018+/_0.005	**0.001**

Fig B. EMG activity (amplitude in mV) in 31 controls and 31 patients during passive movement



Range and velocity of movement:

Table 13 shows the velocity and range of movement in patients and controls during active pronation and supination. It can be seen that the controls had a velocity and range of 487.68 deg/sec and 139.16 deg during active pronation, It was significantly greater than that seen in the patients who had a velocity of 396.29 deg/sec and range of 123.90 deg. (pvalue <0.05). During active supination, it can be seen that only the range of movement was 139.68 deg in the patients and 125.16 deg in the controls (p value 0.018). There was no significant difference during passive movement in both the sets of population since these movements were performed by the examiner.(Table 13).

Table 13. Mean velocity (in degree/sec) and range of movement (in degree) obtained in 31 controls and 31 patients during pronation/supination

	pt status	N	Mean velocity (in degree/sec)	P value	Mean range of movement (in degree)	P value
During pronation (active)	Controls	31	487.68(91.91)	**0.005**	139.16(13.95)	**0.009**
	Patients	31	396.29(149.17)		123.90(28.14)	
During supination (active)	Controls	31	414.97(104.89)	0.725	139.68(14.89)	**0.018**
	Patients	31	427.16(161.49)		125.16(29.86)	
During supination (passive)	Controls	31	454.68(55.13)	0.280	148.45(10.48)	0.485
	Patients	31	436.0(77.97)		150.55(12.91)	
During pronation (passive)	Controls	31	452.81(55.15)	0.746	146.68(9.32)	0.123
	Patients	31	446.48(92.91)		151.16(12.94)	

Post-operative Surface EMG recordings in the pronators and supinators :

The baseline electrical activity in the pronators and supinators at rest was 0.054(0.022) mV and 0.048(0.021) mV respectively. This shows a significant reduction by 21% and 36% from the pre-operative values of 0.069(0.025) mV and 0.076(0.078) mV respectively. ($p<0.05$).

Active pronation and supination

Table 14 shows the mean RMS amplitude in the pronators and supinators during pronation and supination in the patients before and after surgery. It can be seen that the co-contraction of the pronators during supination movement reduced from 0.219 mV to 0.082 mV after surgery – a reduction of almost 62%(p value 0.000). The activity in the supinators during pronation reduced from 0.125 mV to 0.083 mV- a reduction of almost 33% (p value= 0.026). (Table 14)

Table 14. Pre-operative and post-operative mean RMS amplitude in 31 patients in pronators and supinators during active pronation and supination

Movement	**Muscle**	**Mean RMS amplitude (mV) (sd)**		**P value**
		Pre-op	**Post-op**	
Pronation	Pronators	0.795+/_0.487	0.671+/_0.430	0.128
	Supinators	0.125+/_0.088	0.083+/_0.12	**0.026**
Supination	Pronators	0.219+/_0.161	0.082+/_0.08	**0.000**
	Supinators	0.721+/_0.45	0.58+/_0.51	0.066

Passive pronation and supination

Table 15 shows the mean RMS amplitude in the pronators and supinators during passive pronation and supination in the patients before and after surgery. It can be seen that the co-contraction of the pronators during supination movement reduced from 0.491 mV to 0.376 mV after surgery – a reduction of almost 23% (p= 0.007). The activity in supinators during supination decreased from 0.130 mV to 0.084 mV after surgery -- a reduction of almost 35% (p= 0.023) (Table 15).

Table 15. Pre-operative and post-operative mean RMS amplitude in 31 patients in pronators and supinators during passive pronation and supination

Movement	**Muscle**	**Mean RMS amplitude (mV) (sd)**		**P value**
		Pre-op	**Post-op**	
Pronation	Pronators	0.097+/_0.11	0.076+/_0.10	0.082
	Supinators	0.284+/_0.23	0.278+/_0.25	0.835
Supination	Pronators	0.491+/_0.30	0.376+/_0.22	**0.007**
	Supinators	0.130+/_0.17	0.084+/_0.15	**0.023**

Range and velocity of movement: The mean (sd) range of movement produced during active pronation and supination were 123.97(28.07) degrees and 127.39(29.73) degrees. The angular velocity during these movements in the controls were (in degree/second) 376.61(136.93) and 417.32(183.16) for the active pronation and supination. The mean (sd) range of movement produced during passive supination and pronation were 144.58(14.04) and 145.58(13.08) degrees. The angular velocity during these movements were (in degree/second) 432.06(108.21) and 452.61(106.60) for the passive supination and pronation.

Table 16 shows the velocity and range of movement in patients before and after surgery during active and passive pronation and supination.There was significant decrease in the range of movement during passive pronation and supination in the patients after undergoing surgery ($p < 0.01$). However there was no significant change in the velocity of movement during both active and passive pronation and supination.(Table 16)

Table 16. shows the pre- and post-operative mean velocity (in degree/sec) and range of movement (in degree) obtained in 31 patients during pronation/supination

Variable	Pre-operative mean velocity (sd) in degree/second and range (sd) in degree	Post-operative Mean velocity (sd) in degree/second and range (sd) in degree	p value
Velocity during pronation (active)	396.29 (149.18)	376.61 (136.94)	0.386
Range of movement during pronation(active)	123.90 (28.14)	123.97 (28.08)	0.989
Velocity during supination (active)	427.16 (161.49)	417.32 (183.16)	0.679
Range of movement during supination (active)	125.16 (29.86)	127.39 (29.74)	0.661
Velocity during supination (passive)	436.00 (77.97)	432.06 (108.22)	0.776
Range of movement during supination (passive)	**150.55 (12.91)**	**144.58 (14.04)**	**0.010**
Velocity during pronation (passive)	446.48 (92.91)	452.61 (106.60)	0.701
Range of movement during pronation (passive)	**151.16 (12.94)**	**145.58 (13.08)**	**0.004**

Post-operative clinical improvement versus reduction in the co-contraction activity:

The patients were categorised according to the subjective feeling of improvement:

0- no improvement

1- mild improvement

2- good improvement

All patients reported an improvement either mild (20 patients) or good (11 patients). Between these two groups, there was significant decrease in the pronator activity during active supination (p value < 0.05). There was also significant decrease in the pronator activity during passive supination (p value <0.05). There was a decrease in the co-contraction in the antagonists during supination.

Post-operatively, we found that 6 patients remained the same and 15 patients improved in their MAS scores. The remaining 10 patients had normal MAS scores pre-operatively and were excluded from the analysis.

Table 17 shows the pre-operative and post-operative mean RMS amplitude in the pronators and supinators during active pronation and supination in patients. When we compared the EMG activity in those who improved on the MAS score (15 patients) and those who did not (16 patients), we found that there was a significant reduction of co-contraction in the pronators during supination in all the patients, including those whose MAS scores remained the same post-operatively ($p<0.05$). The reduction was 60% in those who remained the same on the MAS score and 52.4% in those who showed an improvement on the MAS score. During passive movement, there was no significant decrease in cocontraction in both the groups.

Table 17. Pre-operative and post-operative mean RMS amplitude in pronators and supinators during active pronation and supination based on improvement in MAS scores.

Movement	Muscle	MAS	Mean RMS amplitude (mV) (sd)		P value
			Pre-op	Post-op	
Pronation	Pronators	Improved	0.710+/-0.422	0.560+/-0.316	0.085
		Same	0.748+/_0.572	0.591+/_0.264	0.356
	Supinators	Improved	0.122+/-0.100	0.104+/-0.17	0.573
		Same	0.11+/-0.102	0.05+/-0.02	0.144
Supination	Pronators	Improved	0.229+/-0.166	0.109+/-0.10	**0.021**
		Same	0.166+/-0.06	0.066+/-0.026	**0.013**
	Supinators	Improved	0.798+/-0.522	0.773+/-0.65	0.830
		Same	0.535+/-0.401	0.401+/-0.274	0.144

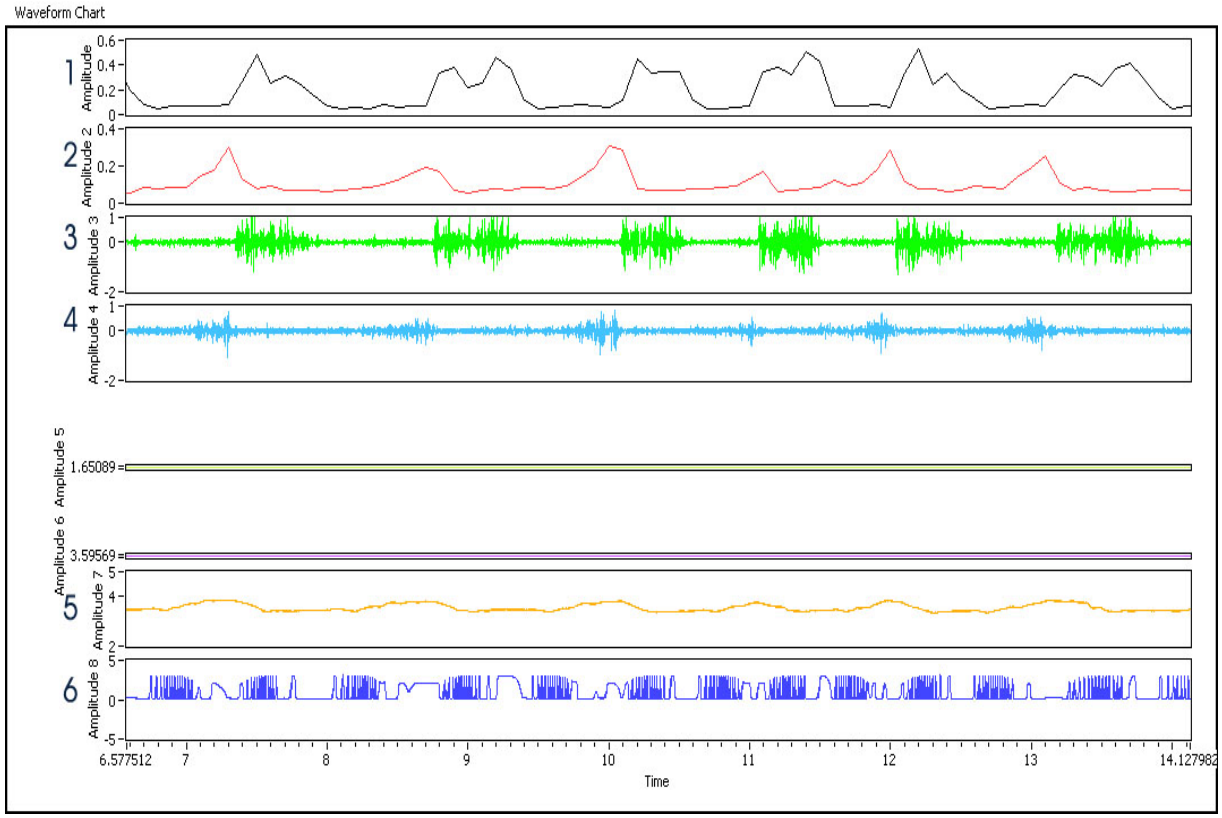
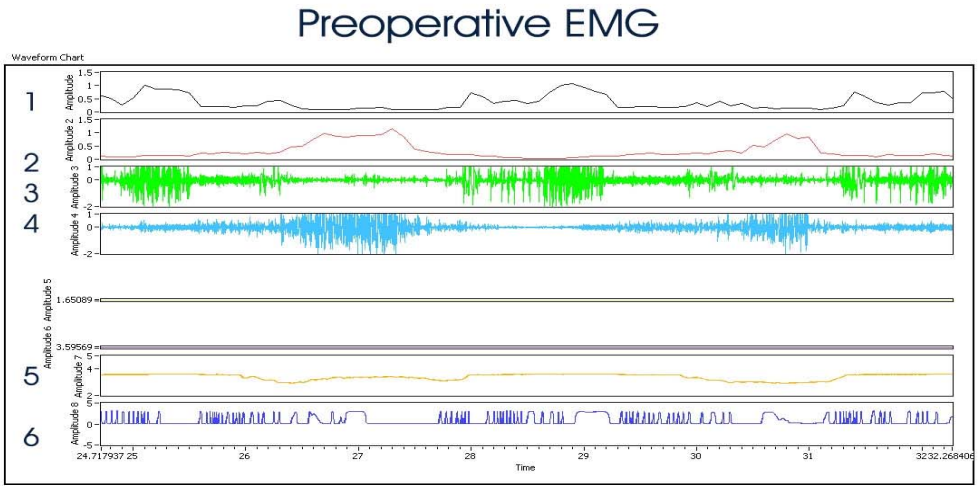


Fig. C. EMG activity in a normal subject

1. RMS EMG Pronators
2. RMS EMG Supinators
3. Raw EMG Pronators
4. Raw EMG Supinators
5. Direction sensor (movement)
6. Position encoder (range and velocity)

We can observe in the above graph (Fig C) that there is more number of contractions with less duration of contraction in the pronators and supinators in the timescale of 7 seconds. There is minimal activity in the antagonistic muscles during agonist activity.



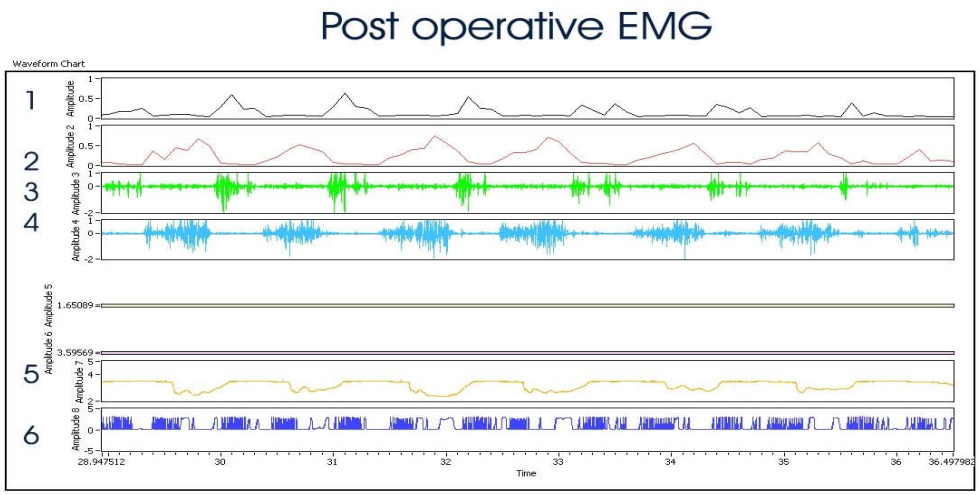


Fig. D. EMG activity in a patient (31)

Legend for Fig. D

1. RMS EMG Pronators
2. RMS EMG Supinators
3. Raw EMG Pronators
4. Raw EMG Supinators
5. Direction sensor (movement)
6. Position encoder (range and velocity)

Figure D shows the EMG recorded in a patient during active pronation and supination in the pronators and supinators. He was symptomatic for 5 years with progressive spastic quadriparesis. His functional grade was Nurick grade 4, MAS score of 1 and JOA score of 6 pre-operatively. He underwent C5 oblique corpectomy. Post-operatively, he had good subjective improvement,, Nurick grade 3 , MAS score was 1 and JOA score improved to 7. The mean RMS amplitude in the pronators during supination reduced from 0.57 mV before surgery to to 0.05 mV after surgery. The mean RMS amplitude in the supinators during pronation reduced from 0.2 mV to 0.05 mV after surgery.

It can be seen that in this timescale of 7 seconds, the pronators contracted thrice and supinator contracted twice. Post-operatively, the patients's alternating movements were much rapid for the same time period when the pronators and supinators contracted seven times each. What is also noted is that the firing is much more prolonged pre-operatively though the instruction was to do the movements as fast as the subject could.

DISCUSSION

In a previous study by Prabhu et al 2003, called the rapid hand flick test (RHFT), complete, rapid opening and closing of the hand was timed for 20 repetitions in patients with cervical spondylotic myelopathy, called the Rapid Hand Flick Time (RHFT), preoperatively and in the first week postoperatively. The results of this test were correlated with the Jebsen-Taylor test. The Jebsen-Taylor test of hand function assesses hand disability and improvement in hand function after therapeutic intervention. There was a 40% to 50% prolongation in the RHFT in patients compared with age-matched normal subjects indicating that there was marked slowing of these movements in patients with cervical spondylotic myelopathy. This was felt to be due to dysregulation of the agonist-antagonist balance of muscle action during flexion and extension movements of the hand. Postoperatively, there was a significant decrease in the RHFT for 20 repetitions indicating an improvement in hand function and this correlated with an improvement in the Jebsen-Taylor test as well as the subjective sense of neurologic improvement reported by most patients post-surgery. This test was thus seen as a reliable and reproducible bedside test of hand function in the immediate postoperative period. It is likely that the slowing of the movement was due to co-contraction in the long flexors of the fingers during extension of the fingers due to spasticity. However, we did not have simultaneous EMG studies to confirm this.

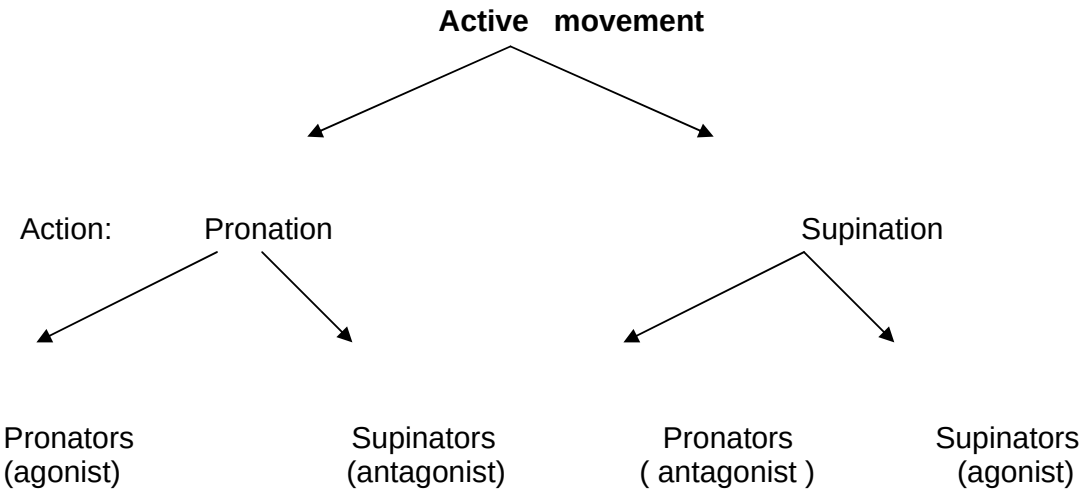
The aim of the present study was to record the surface electromyographic activity from the agonist/antagonist forearm muscles in the upper limbs during alternate pronation/supination and compare the recorded activity with the same post-operatively. We initially attempted to record EMG activity in the flexors and extensors

of the fingers during the RHFT, but had the following technical difficulties: the EMG recordings from flexors and extensors of the hand during opening and closing of the hand required the placement of multiple electrodes and there was no demonstrable silent period observed during agonist/antagonist contraction. Furthermore, accurate placement of electrodes was not possible and the accelerometer placed on the distal phalanges did not give accurate determination of the movement of the fingers. Hence it was decided to study the EMG activity in the pronation and supination of the elbow i.e., pronators and biceps brachii respectively.

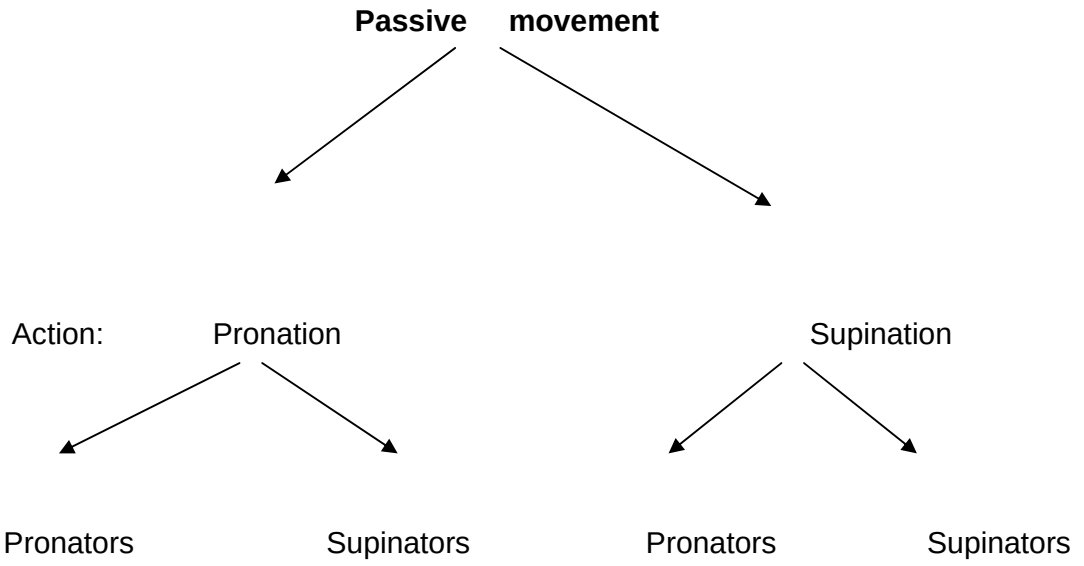
In the clinical assessment of spasticity in patients with myelopathy, the examiner grasps the patient's hand to pronate and supinate the forearm at varying velocities. The feeling of a "catch" while supinating the patient's forearm or a resistance to supination is clinically diagnostic of spasticity (Brennon JB 1959). This is the basis of the modified Ashworth score(MAS) which is currently the widely accepted method of clinically assessing spasticity. But this is a subjective test. We proposed to study this disturbed balance in the upper limb during pronation/supination between the agonist and antagonist muscles by recording surface EMG data.

In our study, the clinical and electrophysiological portions of the study were done in the same session, within 10 minutes of each other, thus limiting the variability. We made efforts to control the testing environment, to separate the subject from any extraneous stimulation. During active movement, the agonists would be showing more activity as they are actively contracting and the antagonists would be relatively silent. During passive movement, there would be less activity in both the group of muscles as there are no agonists and antagonists. We measured

the electrical activity in both the agonists and antagonists of pronation and supination during active and passive movement.



Activity would be more in the agonists than the antagonists during active movement.



Activity would be more in the supinators during pronation and pronators during supination during passive movement due to the stretch produced in the opposite group of muscles.

The data in this study was from age adjusted 31 controls (25 males and 6 females) with a mean age of 53.39 (+/-8.6) years (range 29-62) and 31 patients - 28 (90.3%) males and 3 (9.7%) females with age ranging from 25 to 72 yrs. (mean age of 54.06 yrs± 10.12).

Clinically after the patients underwent surgery, there was a statistically significant change in their functional status in Nurick grade which improved from 3.61 to 3.48 ($p<0.05$), in the Modified Ashworth Score which decreased from 1.97 to 1.45 ($p<0.001$) and upper limb JOA score which improved from 5.45 to 5.94($p<0.001$).

In comparison to the normal subjects, patients had statistically significant increased baseline electrical activity in both the groups of muscles. There was 55% greater baseline activity in the pronators and 61% in the supinators in the patients compared to normal population.

The EMG activity in the agonists during active pronation and supination was higher in patients since they are weaker and recruit more motor units to complete the task. The agonist activity in pronators and supinators during active movement was greater by 24% and 40% respectively. The co-activation noted during active movement was also significantly more in the patients compared to controls by 84% in the pronators during supination and supinators during pronation. Similarly during passive movement, it can be seen that the co-activation of muscle being stretched i.e., supinators stretched during pronation and pronators stretched during supination was significantly higher in patients than the controls-by 43% and 48% for pronators and supinators during supination respectively. We also observed that there was statistically significant decreased velocity and range of movement in the patients during active pronation and supination.

The improvement in the outcome after surgery in our patients was shown by the decrease in the baseline activity in the pronators and supinators by 21% and 36% respectively. The mean amplitude of contraction in the supinators during active pronation and pronators during active supination decreased significantly by 33% and 62% respectively ($p<0.05$). However, during passive supination, the activity in the pronators and supinators decreased significantly by 23% and 35% respectively (p value<0.05).

Lance et al (1980) defined spasticity as a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome. The co-contraction or co-activation which we observed is due to the increased tonic stretch reflex and reflex hyperexcitability, and hence a measure of spasticity. The fact that after surgery, there is a reduction in this , is a good measure of spasticity.

There was a significant reduction of co-contraction in the pronators during supination in all the patients, including those whose MAS scores remained the same post-operatively ($p<0.05$). The reduction was 60% in those who remained the same on the MAS score and 52.4% in those who showed an improvement on the MAS score. The subjective improvement was also considered as an important factor in the analysis. The subjects were grouped according to nil, mild and good subjective feeling of improvement. When the EMG variables were analysed , it was observed that the more the subjects felt improvement, the better the change in cocontraction seen in the EMG. However there is a need for a further followup of the patients who did not have clinical improvement but showed improvement on the EMG parameters.

The tests available for assessment of spasticity are JOA, MAS. Though the patients subjectively felt an improvement, they could not be identified by utilizing the MAS/JOA scores. EMG activity detected the change better as they correlated with the subjective improvement as all patients expressed that they were subjectively better.

We studied the changes in these patients and found a significant difference indicating the objective evidence that surgical decompression leads to a change in the pathogenesis, in that there is probably a shift towards functional recovery.

We presume that the improvement after surgery could be due to the descending excitatory systems becoming functional. Flexor reflexes are probably regained from the inhibitory influences of the dorsal reticulospinal, vestibulospinal and medial reticulospinal tracts.

Engsberg et al had found that there was reduction of elbow flexor spasticity from 0.04 oules/degree/second preoperatively to 0.00 joules/degree/second postoperatively at 11 days. There were less significant results obtained for the ankle dorsi- and plantar flexors. They found that post-operatively improvement was seen as early as 7-11 days. We also observed evidence of improvement based on the surface EMG recordings. As the spasticity affects the antigravity muscles like upper limb flexors, pronators, lower limb extensors and dorsiflexors, our results also showed that there were significant changes in the pronator group of muscles.

Limitations of our study

However, this test had some drawbacks. It was not conducted on consecutive patients, and only considered the symptomatic forearm and the other forearm was not tested. Since the test was conducted at a laboratory it necessitated transfer of patients from the ward to the laboratory. This was discomforting to the patients, since they have difficulty in walking with tightness and flexor spasms. We feel that there is a need to develop patient friendly and portable device to do this testing so as to make it a comfortable experience to the patients. This would need further fine tuning of the device and also a large number of patients to make any further observations and conclusions.

Another feature which could be studied is the measurement of the actual resistance offered to passive movement and observing any decrease in this variable.

This study involved using surface electrodes for obtaining the EMG activity. Though this is non-invasive and simple, we do not know whether needle electrode placement would give a more representative study of the muscle activity.

SUMMARY AND CONCLUSIONS

The present work was undertaken to study the surface electromyography activity in the agonists and antagonists of pronation/supination in the upper limbs in patients with cervical compressive myelopathy and to compare them preoperatively and post-operatively.

A total of 31 patients (age ranging from 25-72 yrs.) with cervical compressive myelopathy were studied.

Surface EMG activity in the agonists and antagonists of pronation/supination in the upper limbs were recorded preoperatively and compared with the recordings obtained in the first week postoperatively by using the pronation-supination apparatus.

The conclusions from the study are:

- The abnormal contraction in the antagonists during active pronation and supination in the patients decreased significantly as a result of the decompressive surgery, more so in the pronator group of muscles during supination.

- The activity in the pronators and supinators during passive supination (and not pronation) in the patients decreased significantly after surgery.

- Whereas MAS and JOA are good clinical measures of spasticity and function, EMG is helpful in the early detection of improvement even in those who did not improve clinically or had mild subjective improvement in the immediate post-operative period.

- There is a need for further follow-up of the patients who have not showed clinical improvement but improved on EMG to observe whether they go on to have clinical improvement.

- However, these results need further confirmation in a larger number of patients.

- Further finer combination of electrophysiological and biomechanical methods will be helpful to study these patients.

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ANNEXURES

CONSENT FORM - (ANNEXURE-I)

CONSENT TO TAKE PART IN A CLINICAL TRIAL

Study Title: Surface electromyography activity in the agonists and antagonists of pronation/supination of the upper limb in patients with cervical compressive myelopathy .

Study Number:

Participant's name:

Date of Birth / Age (in years):

I_____

_____, son/daughter of _____

(Please tick boxes)

Declare that I have read the information sheet provided to me regarding this study and have clarified any doubts that I had. [ ]

I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]

I understand that I will receive free treatment for any study related injury or adverse event but I will not receive and other financial compensation [ ]

I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access [ ]

I understand that my identity will not be revealed in any information released to third parties or published [ ]

I voluntarily agree to take part in this study [ ]

Name:

Signature:

Date:

Name of witness:

Relation to participant:

Date:

ANNEXURE-II

I) Proforma

II) MRC Grading of muscle strength

III) Modified Ashworth Scale for grading spasaticity

IV) JOA score

V) Nurick grading of functional status

(I) PROFORMA

Name Age Sex

Hospital No.

Occupation

Diagnosis :

Duration of symptoms :

Examination of upper limb : Right/Left

Date of examination :

Date of surgery :

CLINICAL EVALUATION

I) Severity of involvement

Wasting of small muscles of hand :

Wasting of forearm muscles :

Nurick's grade :

JOA and upper limb JOA score :

MRC Grading

Shoulder

Elbow

Wrist

Fingergrip

MRI Cervical spine

Level of compression -

PLL Thickening -

Cervical canal stenosis -

Ligamentum flavum thickening -

Cervical spinal cord changes -

Disc prolapse -

Presence of tumor

II)Subjective spasticity score

Modified Ashworth score of upper limb

Pre-operative day

Post-operative day

III) Patient's subjective feeling of improvement

(0)Nil/ Mild(1)/ Good(2)

Experimental results :

PRE-OPERATIVE DAY

Active and Passive movement

Range of Movement deg.

Velocity of Movement deg./sec

EMG

Overlap(co-contraction)

RMS value(root-mean –square)

POST-OPERATIVE DAY

Active and Passive movement

Range of Movement deg.

Velocity of Movement deg./sec

EMG

Overlap(co-contraction)

RMS value(root-mean –square)

(II) The Medical Research Council (MRC) Scale of muscle strength Grades 0-5

0 No contraction

1 A flicker or trace of contraction

2 Active movement with gravity eliminated

3 Active movement against gravity

4- Active movement against gravity and slight resistance

4 Active movement against gravity and moderate resistance

4+ Active movement against gravity and strong resistance

5 Normal power

(III) Modified Ashworth Scale(MAS) for grading spasticity

0. No increase in muscle tone

1. Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of range of motion(ROM) when the affected part(s) is moved in flexion or extension

2. Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder(less than half) of the ROM

3. More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved
4. Considerable increase in muscle tone, passive movement difficult
5. Affected part(s) rigid in flexion or extension

(IV) Japanese Orthopaedic Association (JOA) Score: (normal -18 points)

A. Motor dysfunction of upper extremity

0 unable to move hands

1 unable to eat with spoon, but able to move hands

2 unable to button shirt, but able to eat with spoon

3 able to button shirt with great difficuly

4 able to button shirt with mild difficulty

5 no deficits

B. Motor dysfunction of lower extremity

0 Complete loss of motor and sensory function

1 Sensory preservation without ability to move legs

2 Able to move the legs, but unable to walk

3 Able to walk on flat floor with a walking aid

4 Able to walk up and/or down stairs with handrail

5 Moderate to significant lack of stability but able to walk up and/or downstairs with handrail

6 Mild lack of stability but able to walk with smooth reciprocation, unaided

7 No dysfunction

C. Sensory dysfunction of upper limbs

0 complete loss of hand fuction

1 severe sensory loss or pain

2 mild sensory loss

3 no sensory loss

D. Sphincter dysfunction

0 unable to void

1 marked difficulty in micturition(retention)

2 mild to moderate difficulty in micturition(frequency, hesitation)

3 normal micturition

(V) Nurick Grading of functional status

0 signs or symptoms of root involvement, but without evidence of spinal cord disease.

1 signs of spinal cord disease,but no difficulty in walking.

2 slight difficulty in walking, not preventing full term employment

3 difficulty in walking, preventing full term employment or the ability to do all housework, but, not severe to require someone else's help to walk

4 able to walk with someone else's help or with the aid of a frame

5 bed bound or wheel chair

MASTER CHART (ANNEXURE-III)

GLOSSARY

S.No Serial number

Name

Age in years

Sex 1- male 2;female

Duration of symptoms in months

Patient status Normal -0, patient -1

Nurick grade--1,2,3,4,5

JOA(UL) :(Japanese Orthopaedic Association) (upper limb) score-- 4-8

Modified Ashworth score(MAS)- 0-4

RHFT- rapid hand flick test--20, 40 and 60 - time in seconds to complete these many hand flicks (preop and postop)

PLL : Posterior longitudinal ligament

EMG: Electromyography

CSM: Cervical spondylotic myelopathy

CVJ: Craniovertebral junction

IVDP: Intervertebral disc prolapse

mV: millivolts

RMS: root –mean-square value

Vel: velocity in degree/second

Subj. impr: subjective improvement(0-nil, 1-mild, 2-good)

ROM: range of motion (in degrees)

Surgeries done:

Anterior cervical discoidectomy (single leve)l-1

Anterior cervical discoidectomy (two level) - 2

Oblique corpectomy-3

Central corpectomy-4

Laminectomy-5

CVJsurgery-6

S.no	age	RMS Amplitude(mV										RMS Amplitude(mV) during Passive movement									
		Pre-operative										Post—operative									
		Baselin	baseline	Supination				Pronation				baseline	Baseline	Supination				Pronation			
		Pronato	supinato	pronato	upinator	l.(deg/s	m(degre	upinator	Pronators	(deg/s	n(degr	pronato	supinato	Pronato	upinator	l.(deg/s	m(degre	upinato	pronator	(deg/s	m(degre
1	63	0.103	0.061	0.333	0.056	547	157	0.18	0.034	474	152	0.097	0.043	0.201	0.05	664	147	0.14	0.087	618	152
2	62	0.061	0.046	0.211	0.05	386	128	0.192	0.082	428	133	0.038	0.039	0.429	0.02	471	141	0.1	0.316	521	131
3	36	0.086	0.069	0.72	0.08	554	133	0.16	0.07	655	139	0.038	0.061	0.554	0.078	403	117	0.1	0.07	551	115
4	25	0.05	0.015	0.104	0.104	524	149	0.049	0.049	464	147	0.007	0.003	0.108	0.06	556	133	0.07	0.075	679	128
5	52	0.058	0.034	0.159	0.06	471	161	0.241	0.095	604	165	0.045	0.026	0.18	0.06	562	163	0.13	0.099	612	165
6	46	0.03	0.038	0.159	0.03	468	165	0.236	0.05	580	165	0.027	0.03	0.214	0.05	466	160	0.26	0.04	458	155
7	65	0.023	0.057	0.468	0.03	581	141	0.201	0.03	468	139	0.034	0.046	0.234	0.019	369	136	0.13	0.018	309	139
8	40	0.06	0.04	0.156	0.06	511	157	0.063	0.096	412	147	0.06	0.03	0.584	0.03	592	128	0.33	0.02	506	133
9	55	0.06	0.08	0.075	0.06	422	160	0.164	0.656	380	149	0.043	0.083	0.193	0.07	469	123	0.65	0.553	451	125
10	57	0.08	0.07	0.416	0.08	403	155	0.165	0.07	461	157	0.07	0.068	0.306	0.05	423	139	0.25	0.04	481	152
11	48	0.05	0.05	0.469	0.05	519	163	0.136	0.188	522	160	0.04	0.04	0.42	0.04	475	144	0.13	0.03	504	147
12	66	0.1	0.07	0.548	0.075	353	147	1.008	0.145	342	144	0.08	0.03	0.483	0.04	271	147	0.65	0.016	452	147
13	48	0.06	0.06	0.723	0.08	306	152	0.538	0.07	326	152	0.06	0.04	0.667	0.06	277	154	0.43	0.06	329	152
14	62	0.13	0.12	0.767	0.285	412	160	0.525	0.16	416	163	0.07	0.07	0.212	0.07	323	160	0.4	0.07	327	160
15	63	0.1	0.17	1.028	0.75	245	115	0.81	0.12	269	112	0.1	0.09	0.949	0.869	211	112	1.04	0.09	276	123
16	49	0.07	0.08	0.47	0.07	369	149	0.193	0.07	409	157	0.06	0.05	0.452	0.06	276	123	0.11	0.06	282	131
17	57	0.12	0.11	0.764	0.07	375	160	0.426	0.06	377	157	0.1	0.09	0.345	0.06	391	152	0.2	0.06	345	155
18	58	0.07	0.05	0.308	0.291	309	157	0.196	0.06	318	157	0.06	0.05	0.2	0.05	292	157	0.17	0.05	329	152
19	48	0.08	0.07	0.767	0.285	412	160	0.525	0.16	416	163	0.07	0.07	0.212	0.08	323	160	0.4	0.07	327	160
20	55	0.07	0.1	0.416	0.08	403	155	0.165	0.08	461	157	0.06	0.07	0.306	0.07	423	139	0.25	0.07	481	152
21	65	0.03	0.04	0.3	0.04	468	165	0.236	0.09	580	165	0.03	0.04	0.214	0.03	466	160	0.21	0.03	458	155
22	50	0.056	0.046	0.265	0.05	471	161	0.241	0.095	604	165	0.05	0.04	0.18	0.04	562	163	0.13	0.015	612	165
23	46	0.05	0.06	0.555	0.12	386	128	0.192	0.082	428	133	0.04	0.056	0.429	0.07	471	141	0.1	0.06	521	131
24	55	0.09	0.07	0.416	0.08	486	128	0.165	0.07	348	128	0.08	0.07	0.306	0.07	522	144	0.25	0.06	416	144
25	55	0.07	0.47	0.469	0.05	510	144	0.136	0.06	415	160	0.048	0.04	0.42	0.04	512	152	0.13	0.04	522	152
26	52	0.05	0.04	1.028	0.75	395	152	0.81	0.05	510	152	0.04	0.03	0.949	0.265	423	144	1.04	0.04	505	160
27	57	0.07	0.07	0.47	0.08	452	160	0.193	0.07	350	160	0.07	0.07	0.452	0.07	390	160	0.11	0.07	392	152
28	41	0.11	0.05	0.507	0.05	436	165	0.182	0.025	455	168	0.05	0.03	0.465	0.03	497	157	0.35	0.03	478	155
29	72	0.08	0.07	0.117	0.07	493	149	0.087	0.046	433	141	0.07	0.06	0.065	0.06	463	152	0.08	0.05	369	147
30	66	0.05	0.04	1.269	0.04	381	152	0.212	0.04	399	155	0.04	0.02	0.695	0.04	327	141	0.19	0.03	388	139
31	62	0.05	0.04	0.783	0.07	468	139	0.183	0.05	537	144	0.02	0.02	0.233	0.04	524	133	0.17	0.04	532	139